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# DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

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A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

# GUILLAIN-BARRE SYNDROME*

Review of the Literature; Presentation of Cases

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Since the paper published by Guillain, Barré, and Strohl in 1916[1] in which they described 2 cases of polyneuritis with albumino-cytologic dissociation with a favorable result, many papers have been written in an attempt to clarify the status of this syndrome. Unfortunately almost every author gave his particular modification of the Guillain-Barré syndrome a new name, and as many as thirty appellations have been found in the literature. The following are examples: radiculoneuritis with acellular hyperalbuminosis of cerebrospinal fluid,[2] acute infectious polyneuritis,[3,4,5,6,7] polyradiculoneuritis with albumino-cytologic dissociation,[8] acute ascending paralysis,[9,10] infectious polyneuritis,[11,12,13,14] acute encephalo-myelo-radiculoneuritis,[15] polyneuritis of unknown etiology,[16] acute polyradiculoneuritis,[17] encephalo-myelo-radiculitis,[18,19] infectious neuronitis,[20] polyradiculo-neuritis[21, 22, 23, 24, 25, 26, 27, 28] neuronitis,[29] albumino-cytologic dissociation in cerebrospinal fluid with xanthochromia,[30] polyradiculoneuritis with albuminocytologic dissociation of cerebrospinal fluid,[31] myeloradiculoneuritis with cell protein dissociation,[32] myeloradiculitis,[33] acute polyneuritis,[34] acute infectious meningo-myelo-radiculitis,[35] meningomyelo-encephalitis and meningoradiculomyelitis,[36] polyneuritis with facial diplegia,[37,38] curable polyradiculoneuritis,[39] infective neuronitis,[40,41,42] peripheral neuritis,[43] chronic progressive polyneuritis,[44] polyneuritis,[45,46] acute diffuse polyradiculoneuritis,[47] acute polyneuritis with facial diplegia,[48,49] acute benign infectious myelitis,[50] acute infective polyneuritis,[51,52,53] acute febrile polyneuritis,[54] multiple neuritis,[55] toxic neuronitis of pregnancy,[56] acute polyradiculoneuritis,[57] meningo-encephalo-myelo-neuritis.[58]

In spite of the multiplicity of titles the whole subject "is still in the formative stage and up to this time has hardly advanced beyond the detailed clinical description and possible relationships," to quote the excellent work of Taylor and McDonald[38] published in 1932.

Although it is generally good practice to avoid using a proper name and to use an anatomic term to designate a disease, in this particular syndrome it would be well to abandon the multiple anatomic terms and merely to designate the composite group as the Guillain-Barré syndrome. This was proposed by Lewey,[59] who said, "It is suggested that we retain the designation Guillain-Barré syndrome which has become popular as a general heading for the various forms

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of polyneuropathy and radiculoneuropathy of unknown etiology." Dr. LaSalle Archambault expressed this opinion in a discussion of the paper of Strauss and Rabiner[33] when he said, "To my mind it is rather unfortunate that a new name should be coined every time a fresh group of cases arises, merely on the basis of some minor and frequently inconstant differential feature."

## EVOLUTION OF THE GUILLAIN-BARRE SYNDROME

Osler[60] in 1892 briefly described "acute febrile polyneuritis" and in the discussion mentioned the possibility of paresis of the facial muscles. Laurans[61] in 1908 published a thesis in which he mentioned that Pierson in 1869 discussed the subject of facial diplegia and difficulty with movement of the legs. Laurans collected 18 cases of facial diplegia associated with neuritic signs and symptoms. In 1894 Targowla[62] reviewed a case of polyneuritis with facial diplegia. Mills[63] in 1898 classified one of the toxemias of the central nervous system as multiple neuritis and also proposed the theory of the neuron as a unit in the classification of organic nervous disease. Sinnigar[64] in 1899 and Thomson[65] in 1910 also described cases of polyneuritis with facial diplegia.

In 1907 Sir Farquhar Buzzard[66] classified all the acute paralyses as:

1. Acute poliomyelitis
2. Acute ascending paralysis
3. Landry's paralysis
4. Acute toxic polyneuritis

Acute poliomyelitis is recognized as a well defined entity and will not be described here. Acute ascending paralysis is a less well defined entity and may be only a fulminant variant of acute poliomyelitis or of the Guillain-Barré syndrome. Landry's paralysis, originally described in 1859,[67] has ceased to be regarded as an entity by most authors,[38,48] as predicted by Walton[68] in 1895. On this subject Grinker[69] states, "Landry's ascending paralysis is not the name of a disease but constitutes a syndrome caused by a number of pathological entities. Anterior poliomyelitis is probably the most common cause. The pathology is dependent on the cause."

Acute toxic polyneuritis was described by Buzzard[66] in 1907 as follows:

1. Light constitutional symptoms, if any.
2. Sensory loss, glove and stocking type, if any.
3. Motor involvement, limbs more affected than trunk, peripheral parts of limbs more than proximal. Diaphragm often involved.
4. Cranial nerves, chiefly facial, sometimes palate is affected.
5. Deep reflexes absent.
6. Abdominal reflexes often retained.
7. Sphincter reflexes, transient involvement occasionally.
8. Relapses frequent.

This was a classical picture of what later became more widely known as the Guillain-Barré syndrome.

An excellent discussion of the causes of multiple neuritis was published by Oppenheim[70] in 1910. In their original paper in 1916 Guillain, Barré, and Strohl emphasized the albumino-cytologic dissociation with a favorable prognosis in 2 cases. They described the condition as a "syndrome characterized by motor disturbances, abolition of tendon reflexes with preservation of cutaneous reflexes, of paresthesias with minor disturbances of objective sensibility, of pain on pressure of muscles and of albumino-cytological dissociation of cerebrospinal fluid." Their report of the dissociation was a definite milestone, but this same discordance between protein and cell count led Guillain into an error in prognosis which was not rectified in his own mind until 1937.

In the same year Patrick[71] carried on the work of Laurans with facial diplegia in multiple neuritis. He collected a total of 48 cases, including 1 fatal case. Cerebrospinal fluid studies were not included in his work. At about the same time J. Hatori and Shoji[46] reported an epidemic of polyneuritis (not beriberi) on the islands of Botel, Tobago, and Formosa with a high mortality. Paralysis of facial nerves was noted, but cerebrospinal fluid studies were not reported.

World War I produced a polyneuritis observed and studied by Bradford, Bashford, and Wilson.[51] They described an initial illness, a latent period and a paralytic stage characterized by:

1. Sensory phenomena, glove and stocking acral type of paresthesias.
2. Reflexes, deep lost, superficial usually retained.
3. Sphincters, not constant.
4. Cerebrospinal fluid, no change in cells. No increased pressure. Protein not reported.

Of their 30 cases, 8 were fatal. Their study of the virus causative agent will be discussed under etiology. Casamajor[9] reported similar cases and mentioned occasional seventh nerve palsy and also negative cerebrospinal fluid. Foster Kennedy[41] also reported a similar condition among troops with 1 fatality out of 4 cases. Holmes[54] studied the same type of syndrome among soldiers.

In 1922 Harris[72] described toxic polyneuritis and drew attention to a case of puerperal neuritis with complete recovery in four months. Skoog[55] in 1924 reported a case with paresis of arms and legs, absence of deep reflexes, bilateral paresis of seventh nerve, and increased globulin in cerebrospinal fluid. He stated, "I wish to call your attention to a severe type of multiple neuritis, moderate amount of myelitis and a lesser encephalitis." In the same year Mettel[53] reported a similar condition in children and postulated that mortality was low in young people. Govaerts[73] reported a case with massive hyperalbuminosis of cerebrospinal fluid and xanthochromia.

Strauss and Rabiner[33] in 1930 reported a series of cases of "myeloradiculitis," all with favorable results. No cerebrospinal fluid studies were reported. In 1931 Sands[50] reported 4 cases of cerebrospinal fluid albumino-cytologic dissociation, all with favorable results. He stated, "It is not at all impossible that this (syndrome) may represent an unusual form of epidemic encephalitis."

In 1932 Collier[43] divided his cases of peripheral neuritis into two types:

1. Four-limb peripheral neuritis with external ophthalmoplegia.
2. Four-limb polyneuritis of fairly rapid onset associated with facial paralysis and often with bulbar paralysis.

He postulated that the cerebrospinal fluid was usually unchanged but occasionally was yellow and contained mononuclear cells.

In 1932 Taylor and McDonald[38] reported 16 cases of polyneuritis with facial diplegia. Their outstanding contribution was the statement, fully as true today as fifteen years ago, "That there is at present no justification for regarding any of these acute infections (Guillain-Barré type) as individual disease entities is furthermore sufficiently demonstrated by the transitional forms and consequent confused clinical picture. The tendency to magnify similarities and minimize differences is well exemplified in this group of disorders in the attempt to establish circumscribed clinical entities."

In 1933 Lubin[56] reported a case of neuronitis of pregnancy associated with vomiting, and in 1934 Maisel[74] a similar case without vomiting. In the same year Schneider[35] reported a case of cerebrospinal fluid dissociation and anemia in which an erroneous diagnosis of posterolateral sclerosis was made. The patient made an excellent recovery in one year. He emphasized the danger of making a diagnosis of posterolateral sclerosis without achlorhydria.

In 1936 Guillain[2] defended his original position and insisted on albumino-cytologic dissociation ("cases with .3-.4 Gm. [300-400 mg. per cent] do not belong to this syndrome") and a favorable prognosis. He also acknowledged the presence of facial palsy in some cases. In the same year Gilpin, Moersch, and Kernohan[45] reviewed cases observed at the Mayo Clinic. About the same time Bassoe[36] reported 3 cases, 1 with papilledema and residuals of ankle clonus, strabismus, and a positive Babinski reflex. Guillain[75] pointed out the danger of making an erroneous diagnosis of tabes because of the persistence of loss of tendon reflexes in his syndrome.

In 1937 Guillain, in a discussion of a paper by Barré,[76] retracted his firm stand that the total protein had to be 1 to 2 Gm. (1000-2000 mg. per cent) for a diagnosis of his syndrome. He also admitted that fatal cases did occur. Van Gehuchten in 1938 presented 1 case with papilledema.[77]

McIntyre[42] reviewed 8 cases and postulated four types:

1. Mild course with rapid recovery.
2. Prolonged course with death from heart failure.
3. Prolonged course with incomplete recovery.
4. Prolonged course with death from bulbar or respiratory paralysis.

He also pointed out similarity of this clinical picture to that of diphtheritic paralysis. Hecht[52] in his review of 8 cases postulated recovery without permanent paralysis unless death results from respiratory paralysis. Guillain and Kreis[78] reported a case with cranial nerves involved exclusively.

In 1938 Garvey and Slavin[6] reported several cases including 1 in a diabetic with bilateral internal and external ophthalmoplegia with recovery. Cossa *et al.*[39] reported a case with absent tendon reflexes and a unilateral Babinski sign.

Barber[23] in 1940 observed that the condition is most often seen in autumn and winter. Shasken *et al.*[32] reported 2 cases in negroes. Garvey *et al.*[25] reported 6 cases following fever therapy. (It should be pointed out that hundreds of others received the same treatment without developing the syndrome.) Stone and Aldrich[57] emphasized the possibility of the late appearance of the cerebrospinal fluid dissociation. De Jong[79] reviewed 15 cases all with dissociation with 2 fatalities. He suggested three groups:

1. With short duration and favorable prognosis.
2. With prolonged or chronic course.
3. With fatal termination.

In 1941 Casamajor and Alpert[80] found 19 cases under the age of 12 in the French literature and a like number in the English literature.

Polan and Baker[18] in 1942 stated, "Deep reflexes as a rule are reduced or absent but may be hyperactive and associated with sustained or unsustained clonus." De Sanctis and Green[5] gave an excellent differential diagnosis between the Gullain-Barré syndrome and poliomyelitis. Spector[81] reported a case with ataxia as the presenting symptom. Glen[27] presented a case with no sensory involvement. Fox and O'Connor[20] reviewed 126 cases, 41 of which had a history of preceding illness, 42 had facial palsy, and 26 terminated fatally.

In 1943 Shaskan[82] pointed out the danger of death from respiratory paralysis. Ford and Walsh[4] reported 2 cases with increased intracranial pressure and papilledema.

Baker[19] proposed five subdivisions of this syndrome: (1) abortive, (2) polyneuritic, (3) myelitic, (4) bulbar, and (5) cerebral.

Hatoff[16] in 1944 stated that the greatest number of cases occurred in July, August, and September.

## ETIOLOGY

Wilson[51] in 1919 believed he had isolated and cultivated the virus of this syndrome. It was postulated that the disease could be transmitted from man to monkey, from monkey to monkey, and the characteristic lesion produced in the animal. However, in the same year after a criticism of his work by Arkwright,[83] Wilson retracted his claim to isolating the virus. In 1932 Collier[43] observed, "The causal relation of virus infection to polyneuritis remains unproven."

In 1938 Barré,[84] commenting on the enormous vasodilatation of the capillaries of nerves and ganglia reported by Balduzzi,[85] said, "We (Guillain and Barré) thought this disease was congestive not inflammatory. This fact explains the good prognosis of the syndrome, the relative rapid return to normal, and leads one to think the cause is still a virus or poison with action vasodilatory and temporary."

In 1941 Sabin and Aring[12] made an attempt to reproduce the work of Bashford and Wilson. Jervis and Strassburger[14] in 1943 advanced the hypothesis that a number of cases of Guillain-Barré syndrome may actually be cases of anterior poliomyelitis with favorable outcome.

The fact that diseases such as diphtheria, syphilis, diabetes, toxemia of pregnancy, serum sickness, mustard gas in isolated instances, may present a clinical syndrome identical with that of the Guillain-Barré syndrome (even including the cerebrospinal fluid albumino-cytologic dissociation) would seem to point toward a toxic agent as the etiologic factor.[42]

## PATHOLOGY

There is a wealth of knowledge concerning the pathologic changes in this syndrome. That the descriptions of various authors

vary widely testifies to the bizarre type of lesions of this syndrome. Holmes[54] in 1917 reported chromatolysis and myelin degeneration. Bradford *et al.*[51] reported:

1. Acute exudative neuritis.
2. Involvement of posterior root ganglia.
3. Degeneration of nerve cells.
4. General involvement of gray matter of brain and medulla.
5. Muscular degeneration.
6. Hyperplasia of lymph glands, congestion at bases of papillae of kidneys, infiltration of round cells in large and small portal tracts of liver. (Aring[12] also observed pathology in organs other than the central nervous system.)
7. Round cell infiltration around damaged or degenerated nerve cells.
8. Hemorrhages throughout cord.

Casamajor[9] reported in 1919, "The nerve fibers show marked degeneration of a toxic nature" described as an increase of neuroglia cells, degeneration of nerve cells, neuronaphagia, and small round cell infiltration. Kennedy[41] in 1919 quoted work of Wilson and described patchy neuritis of peripheral nerves, degeneration of cells in ventral dorsal cornua, cerebral cortex, and pontine nuclei, small round cell infiltration around ganglion cells but never around meningeal vessels (as in poliomyelitis), normal meninges, and proliferation of the ependyma of the central canal.

Hyland *et al.*[44] in 1930 described a normal brain, severe demyelinization of the anterior nerve roots of the peripheral nerves and of the intramuscular nerves, dilatation of the perineural lymphatics, proliferation of the interstitial tissue, and infiltration by mononuclear cells of all the peripheral nerves. Jacobi[40] (1931) reported degeneration of cells of the anterior horn, nerve fibers, cells of the spinal ganglion and the sensory neurons of the tracts of Goll and Burdach, no changes in the dura or pia, decided reduction in Purkinje cells and degeneration in those present, diffuse slight small round cell infiltration in the cerebrum, pons, and subthalamic region, and decrease in the cerebellar cells.

Taylor and McDonald[38] in 1932 pointed out that the findings in the Guillain-Barré syndrome bear a much closer relationship to those occurring in epidemic encephalitis than had hitherto been supposed.

Gilpin *et al.*[45] in 1936 listed the pathologic changes as a patchy degeneration of myelin and fragmentation of the axis cylinders of the peripheral nerves without evidence of inflammation. In 1 case they found inflammatory changes in the dorsal root ganglia and slight ascending changes in the spinal cord. Alajouanine *et al.*[86] reported that all the peripheral, spinal, and cranial nerves were injured with the maximum of the lesion at the level of the roots. The lesions consist of diffuse interstitial lymphocytic infiltration, proliferation and swelling of the cells of Schwann, associated with beginning degeneration of myelin. "The absolute integrity of the cells of the peripheral neurons and also of the motor cells of the anterior horn and the sensory cells of the spinal ganglia give a good evidence of the affinity of the virus (?) for the cells of Schwann and the myelin."

In a discussion of a paper by Faure-Beaulieu and Feld[84] in 1938 Barré quoted Balduzzi's[85] finding of enormous vasodilation of the capillaries of the nerves and ganglia. Stevenson[32] in 1940 noted a pronounced peripheral neuritis, especially in the sciatic nerves, and degeneration of the columns of Goll and Flechsig. He also found no evidence of an acute inflammatory lesion in the peripheral or cranial nerves or in any part of the brain examined. DeJong[79] described degenerative changes in motor nuclei of the anterior cornual cells, consisting of shrinking and pyknosis in some and accumulation of fat in others, with occasional edema and vacuolization. No inflammatory changes and no demyelinization were noted. The nuclei of the medulla showed changes similar to those seen in the anterior horns.

In 1941 Sabin and Aring[12] published a comprehensive study of observations in this syndrome:

PERIPHERAL NERVES
usually degeneration of axis cylinders and myelin sheaths with proliferation of cells of the sheath of Schwann and interstitial infiltration with various inflammatory cells.

ADRENALS
focal degeneration and infiltration with mononuclear cells.

LIVER
focal cellular infiltration in capsule and portal

spaces, focal necrosis of liver cells with cellular infiltration and focal fatty degeneration.

KIDNEYS

focal intertubular infiltration with mononuclear cells.

HEART

interstitial infiltration with mononuclear cells and polymorphonuclear cells and in 1 case necrosis of isolated muscle fibers and focal phlebitis. (This focal phlebitis was also noted in the adrenals and lungs of the same case.)

Polan and Baker[18] in 1942 observed, "The pathologic lesions present no specific features and can be observed in many other nervous system involvements." Baker,[19] however, in 1943 described perivascular foci of demyelinization scattered throughout cerebral hemispheres, neuronal alteration within nuclei of cranial nerves, and patchy areas of myelin destruction within the peripheral nerves. Russell and Moore[87] in the same year reported a chronic case with residual symptoms resulting in death from intestinal obstruction. This case presented no brain lesion, but there was a loss of ganglion cells in the posterior root ganglia with degeneration of the posterior columns of the spinal cord, the posterior nerve roots, and the peripheral nerves without inflammatory reaction.

Jervis and Strassburger[14] in 1943 cited a typical patient with this syndrome who died six months later of bilateral lobar pneumonia. At autopsy they found localization of the process to the anterior horns of the spinal cord and morphologic features of poliomyelitis. The destruction of myelin and nerve cells was generally predominant over the vascular component. Of considerable interest was the almost complete absence of meningeal infiltration, a condition which may account for the low cell count. The breakdown of myelin was perhaps more severe than is generally seen in poliomyelitis.

In 1944 Hatoff[16] reported a rapidly fatal case with cerebrospinal fluid dissociation which at autopsy presented only terminal hyperemia and small hemorrhages in the cord. Lewey[59] in 1945 described acute radiculopathy progressing almost to necrosis. The myelin was pulverized and in many instances completely dissolved and the axis cylinders fragmented. There was no infiltration, no compound granular bodies, and only scant scar formation from the endoneurium.

An analysis of the foregoing observations reveals no common denominator. The most constant change was a degenerative and demyelinating process without signs of inflammation. Small round cell infiltration was next in frequency. The preceding descriptions give a fairly broad idea of what patterns may be expected from necropsy study of this syndrome. The diversity of the pathologic changes further attests the sound judgment of referring to this condition as a syndrome rather than as an entity. Further observation and study may reveal the precise nature of the disease.

## LABORATORY DATA

In this syndrome studies of the cerebrospinal fluid are the most characteristic and constant. Guillaine *et al.*[1] originally pointed out the albumino-cytologic dissociation in 1916. That this was not necessarily constant was evident from the work of Bradford, Bashford, and Wilson,[51] and the studies of Casamajor.[9] In this review of the literature the finding of albumino-cytologic dissociation is reported in about 60 to 80 percent of cases. Baker states,[19] "The absence of a high total protein may be found in typical cases. The presence of mild or moderate cell increase (in cerebrospinal fluid) chiefly mononuclears, is not untenable with this disease (encephalo-myelo-radiculitis)." The cerebrospinal fluid dissociation may be a late manifestation.[57] Xanthochromia is present in a small percentage of cases. In 1 case Taylor and McDonald[38] reported a total protein in the cisternal fluid of 49 mg. per cent and 206 mg. percent in the lumbar spinal fluid. They stated that "further detailed study of the serology of the cerebrospinal fluid from the lumbar, cisternal, and ventricular regions should yield results of importance from a diagnostic standpoint." In this review of the literature no such report was found.

Increased intracranial pressure with papilledema has been reported only rarely with this condition.[4,36] The colloidal gold curve is not characteristic. Sugar and chloride stud-

ies are usually within normal limits. One case[88] was reported with temporary spinal cord block of unknown origin.

The blood and urine studies are not significant. A transient leukocytosis is occasionally seen in the early paralytic stage. Anemia is not a constant observation. Baker[19] pointed out that the heterophile antibody test was positive in many successive cases. An elevated blood sedimentation rate has been reported.[28]

Study of chronaxia[84] has not proved significant.

## CLINICAL COURSE AND SYMPTOMATOLOGY

The occurrence of this syndrome in late summer, autumn, and winter[16,23] is probably only coincidental. The age and sex incidence is apparently not significant. In 33 to 55 percent of cases there is a history of a preceding illness, usually an upper respiratory infection. This illness is often followed by a latent period lasting from a few days to a few months.

The immediate prodromes vary from rather severe headache, backache, ataxia, to mild paresthesias of the hands and feet.

The paresis or paralysis itself is generally symmetrical, equal and of the flaccid type, although Baker[16,18,19] points out that rarely a spastic type is seen. The paresis usually begins in the lower extremities and may spread to the upper extremities. Authors disagree on whether the proximal or distal groups of muscles are more involved.[52,66,71]

The trunk is usually less affected than the limbs. Occasionally the diaphragm is involved. Tenderness of the muscles or over the nerve trunks is noted occasionally. The electric reaction is that of degeneration, more frequently incomplete, the changes being most pronounced in muscles of the extremities.[24]

The sensory involvement tends to be minimal and of the glove and stocking or acral type of distribution. The deep sensory observations are neither constant nor characteristic. In general the deep reflexes are lost and the superficial reflexes are retained. The anal and vesical sphincters may or may not be involved.

The cranial nerves may be affected. The seventh nerves are most frequently involved. This paralysis is usually described as peripheral in type.[2] The actual incidence of facial diplegia is problematic since many authors choose to classify the cases with facial diplegia in a separate category. A few cases[78] present involvement of the cranial nerves exclusively.

The temperature is normal in most cases. Tachycardia[22] has been described but is at best an inconstant finding. The status of the patient varies from the completely helpless patient to one with a mild peripheral neuritis. The course varies from rapid recovery with no residual symptoms to that of a fatal outcome. Often patients make a complete recovery over a period of months. The deep reflexes tend to return slowly.[75] The incidence of contractures is low.[3] Paresis of the facial muscles may persist for years. Relapses or recurrences are not common.

Fatalities are commonly caused by respiratory or bulbar paralysis. Patients with prolonged courses may die of heart failure[42] or terminal pneumonia.

The prognosis is apparently impossible to predict in any given case and should be guarded. Guillain originally felt that in the presence of cerebrospinal fluid dissociation the prognosis was consistently favorable. As previously noted this stand by Guillain was untenable. Mortality varies from 14 percent[46] to 42 percent.[38] Polan and Baker[18] warned that "a prognosis should be made only after a long period of observation."

## TREATMENT

Guillain[2] used intravenous sodium salicylate and intramuscular quinine, methenamine, and colloidal silver. He also used massage with colloidal silver. Barker[47] advocated the removal of focal infections.

Sahs and Paul[29] reported the use of the Kenny treatment in 8 cases and noted that the treatment did not forestall paralysis in the progressive phase. Gayle *et al.*[89] also used Kenny packs. Vitamins, typhoid shock,[33] x-ray, and arsenicals have been tried. Recently the use of neostigmine has been advocated.[90,91]

No treatment has been particularly successful. General supportive measures and the use of a respirator in those cases developing respiratory paralysis are the procedures of choice.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis from anterior poliomyelitis is made by DeSanctis and Green[5] as shown in Table I.

The differential diagnosis of Guillain-Barré's syndrome from diphtheria means only the recognition of diphtheria as a possible associated condition, since clinically the two cannot be separated.[42] Likewise, cases of polyneuritis with or without facial diplegia in diabetes, in syphilis, in severe deficiency disease, and in pregnancy or in the puerperium cannot be differentiated clinically from those of unknown etiology. An interesting feature is that this syndrome may develop in syphilitics under adequate treatment, in controlled diabetics, in gravid women with no toxemia,[74] and in women in the puerperium who have had normal pregnancies and deliveries.[72] On the contrary, untreated syphilitics who develop this syndrome may clear up completely from a neurologic aspect on antileutic therapy.[92, 93]

Acute disseminated encephalomyelitis with extensive root symptoms may simulate the clinical picture. This disease is not discussed because some authors, such as Wilson and Bruce,[94] doubt the existence of this entity. Certainly some of the cases described by Stout and Karnosh[95] as acute disseminated encephalomyelitis of the peripheral polyneuritic type could easily have been classified under the Guillain-Barré syndrome. In their cases albumino-cytologic dissociation was frequent, though not invariably seen. Pyramidal signs are more commonly described in disseminated encephalo-myelitis than in Guillain-Barré syndrome.

The toxic neuritides from alcohol and other exogenous poisons such as metals may usually be differentiated from the history and peripheral character of the disease.

Another group of cases of polyneuritis with and without facial diplegia, often with albumino-cytologic dissociation, is seen following serum sickness[96, 97, 98, 99] and following exposure to mustard gas.[100] A diagnosis may be made easily from the history, although clinically the syndromes are identical.

## PRESENTATION OF CASES

During the past nine years 15 typical cases of the Guillain-Barré syndrome have been recognized at the Cleveland Clinic. Three other cases are presented because of their clinical similarity. The summary of

TABLE I

*Differential Diagnosis of Acute Infectious Polyneuritis (Guillain-Barré) Syndrome and Acute Anterior Poliomyelitis.*

| | *Acute Infectious Polyneuritis* (Guillain-Barré Syndrome) | *Acute Anterior Poliomyelitis* |
|---|---|---|
| HISTORY | Usually slow, progressive, but may be sudden. | Slow progressive onset. |
| PAIN | Hyperesthesia and muscle pain. | Muscle pain. |
| INVOLVEMENT | 1. Ascending paralysis.<br>2. Bilateral, symmetrical involvement, usually beginning in proximal groups of muscles of extremities.<br>3. Cranial nerve involvement usually affects only seventh nerve. | 1. Ascending paralysis.<br>2. Segmental, often unilateral with irregular muscular involvement.<br>3. Cranial nerve involvement; multiple cranial nerves involved. |
| PROGRESS | New paralysis may develop during a prolonged period after onset. | No further paralytic involvement after two weeks from onset. |
| LABORATORY | Albuminocytologic dissociation in spinal fluid. | Cellular increase in spinal fluid, early polymorphonuclears, followed by lymphocytes. |
| PROGNOSIS | Good. | Guarded for a prolonged period. |

## TABLE II

*Guillain-Barré Syndrome*
*Summary of Cases*

| Case | Age | Sex | Onset | Preceding Infection | Location Initial Symptoms | Cranial Nerve Involvement | Sensory Changes | Deep Reflexes | Sphincter Disturbance | Cerebrospinal Fluid: Total Protein mg. per cent | Cerebrospinal Fluid: Cells per cu. mm. | Cerebrospinal Fluid: Xanthochromia | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 52 | M | April, 1946 | 0 | Cranial nerves | VII | + | Absent | + | 105 | 0 | 0 | Satisfactory improvement in 5 mo. |
| 2 | 23 | F | Mar., 1939 | 0 | Cranial nerves | III, IV, VI VII, VIII | + | Absent | 0 | 455 | 0 | + | Death in 4½ yrs. of terminal pneumonia |
| 3 | 21 | F | July, 1939 | 0 | Arms | 0 | + | Absent | 0 | 288 | 0 | 0 | Recovery in 5 mo. |
| 4 | 23 | M | Mar., 1940 | + | Legs, arms, cranial nerves | VII | + | Absent | 0 | 240 | 0 | 0 | Recovery in 5 mo. Residual facial palsy present 6 yrs. later |
| 5 | 51 | M | Sept., 1940 | + | Legs and arms | 0 | + | Absent | 0 | 70 | 1 | 0 | Recovery in 5 mo. |
| 6 | 48 | M | Feb., 1941 | + | Legs and arms | 0 | + | Absent | 0 | 120 | 0 | 0 | Recovery in 3 mo. Mild recurrence 4 yrs. later |
| 7 | 35 | F | Feb., 1942 | + | Legs and arms | 0 | + | Absent in legs, diminished in arms | 0 | 65 | 0 | 0 | Recovery in 11 mo. |
| 8 | 35 | M | April, 1942 | 0 | Legs | 0 | + | Absent | 0 | 415 | 0 | 0 | Recovery in 5 mo. |
| 9 | 56 | M | June, 1943 | + | Legs and arms | 0 | + | Absent | 0 | 120 | 0 | 0 | Recovery in 10 mo. |
| 10 | 35 | F | Feb., 1945 | + | Legs and arms | 0 | + | Diminished | 0 | 120 | 2 | 0 | Death in 11 mo. of acute necrotizing hepatitis |
| 11 | 19 | F | April, 1945 | 0 | Legs and arms | VII | 0 | Absent | + | 58 | 1 | 0 | Recovery in 4 mo. |
| 12 | 54 | M | June, 1943 | 0 | Legs and arms | VI(right) | 0 | Absent | + | 385 | 8 | + | Death in 6 mo. of terminal pneumonia |
| 13 | 21 | M | Feb., 1946 | 0 | Legs and arms | 0 | + | Absent | 0 | 350 | 0 | + | Recovery in 7 mo. |
| 14 | 52 | M | Jan., 1941 | 0 | Legs | 0 | + | Absent in legs | 0 | 110 | 0 | 0 | Death in 11 mo. of cardiac failure |
| 15 | 53 | M | Dec., 1937 | 0 | Legs and arms | VII | 0 | Absent | 0 | ++++ Globulin | 0 | 0 | Death in 4 wks. of respiratory failure |
| * 16 | 26 | F | May, 1942 | Pregnancy | Legs and cranial nerves | VII(left) | + | Absent | 0 | 470 | 2 | + | See analysis of cases |
| * 17 | 51 | F | Feb., 1940 | Diabetic | Dorsum | 0 | + | Diminished in legs | 0 | 85 | 0 | 0 | No improvement in 6 mo. |
| * 18 | 66 | F | July, 1940 | Diabetic | Legs | 0 | + | Diminished in legs | 0 | 270 | 7 | 0 | No follow-up study |

*Included as examples of clinically identical picture seen in other conditions.

these cases is presented in Table II. Several cases clinically diagnosed as the Guillain-Barré syndrome were excluded from the study because of the absence of albuminocytologic dissociation.

## ANALYSIS OF CASES

The first 15 cases presented albuminocytologic dissociation of unknown origin. This group of cases showed the following:

*Preceding Infection.* Six cases gave a history of an upper respiratory infection. The interval between the infection and onset of symptoms varied from two weeks to three months.

*Age, Sex, and Seasonal Incidence.* The age incidence was highest in the sixth decade, in which 6 of the cases occurred. Four cases occurred in the third decade. The sex incidence was 10 in males, 5 in females. The seasonal incidence was highest in February, in which 4 cases had their onset. Three cases originated in April.

*Initial Onset.* In 9 cases the initial symptoms, mainly of weakness and paresthesias, were in the arms and legs. In 2 cases the initial symptoms were in the legs. In 2 cases involvement of a cranial nerve was the presenting symptom. In 1 case the presenting symptoms were in the arms, and in 1 case the arms, legs, and cranial nerves were involved initially.

*Cranial Nerve Involvement and Sphincter Disturbance.* The cranial nerves were involved in 6 instances. In 4 cases there was bilateral facial diplegia. In 1 case bilateral facial diplegia was seen in combination with involvement of the third, fourth, sixth, and eighth nerves. This case had a complete external ophthalmoplegia, an uncommon finding in this syndrome.[6,19,43] A right sixth nerve palsy was observed in 1 case. Sphincter disturbances were present in 3 cases, namely urinary retention in 2 instances and fecal incontinence in the third.

*Temperature and Pulse.* The temperature in all cases was within normal limits. The pulse rate was over 100 in 6 cases.

*Motor and Sensory Involvement.* The most common observation was a motor weakness of the legs. The sensory involvement was most common in the legs, and chiefly of the glove and stocking type of acral anesthesia. In 3 instances no sensory loss was noted. In 2 cases the sensory loss was confined to ulnar distribution of the hands. Vibratory and position sense were diminished in the legs in 6 instances. The nerve trunks were tender in 1 case. In only 1 instance was respiratory failure observed in the acute phase.

*Reflexes.* The deep reflexes were absent in 12 cases. In 1 case the deep reflexes were absent in the legs only, in another they were absent in the legs and diminished in the arms. In 1 case all deep reflexes were diminished. In most instances the abdominal reflexes were preserved.

*Laboratory Study.* Hemoglobin was below 70 percent in only 1 instance. The highest leukocyte count observed was 11,950. Small amounts of albumin were found in the urine in 4 cases. The cerebrospinal fluids varied from a total protein of 58 mg. percent to 455 mg. percent. The cell counts varied from 0 to 8 cells per cu. mm. Xanthochromia was observed in 3 cases. The colloidal gold curve studies were not significant. The Wassermann reactions were negative in the spinal fluid and blood in every case. Case 11 was seen in the Cleveland Clinic Hospital on April 17, 1945, nine days after the onset of initial symptoms, at which time the cerebrospinal fluid presented a total protein of 58 mg. percent and 1 cell per cu. mm. Subsequently this patient was followed at the Henry Ford Hospital in Detroit, and the cerebrospinal fluid studies there in June, 1945, revealed a total protein of 425 mg. percent and 1 cell per cu. mm. The fact that cerebrospinal protein may not be elevated early in this syndrome has been previously described.[57]

## TREATMENT

Various forms of treatment were used including deep x-ray therapy over the dorsal spine, typhoid shock, and arsenical therapy. Vitamins, chiefly B complex, and liver extract were also used. Therapy had little if any effect on the clinical course.

## RESULTS

Five of the cases terminated fatally. One patient died of respiratory failure four weeks after the onset of initial symptoms. One died of terminal pneumonia after six months without improvement. One died of cardiac failure after nine months of continuous illness. One patient died of necrotizing hepatitis after eleven months. The fifth patient lingered on for almost five years without improvement and then succumbed to terminal pneumonia. The ages of these patients were 54, 53, 52, 35, and 23 respectively. The sex incidence was 3 males and 2 females. In only 1 case was a previous history of infection elicited. Xanthochromia of the cerebrospinal fluid was present in 2 of the fatal cases.

In 7 cases there was complete recovery except for absence of tendon reflexes in 2 instances from three to ten months after onset. One patient was improving satisfactorily five months after onset of initial symptoms. The only residual signs were slight facial palsy (still present six years after complete recovery otherwise) in 1 case and slight weakness of the legs in a second case. In case 6 a mild recurrence of the original symptoms occurred four years after original illness and cleared up completely in a few months.

*Necropsy Study.* The necropsy study of case 10 is included through the cooperation of Dr. H. K. Giffen of Youngstown, Ohio. The significant findings were:

1. Focal degeneration in and around the spinal cord.
2. Edema of the brain with hyperemia.
3. Degeneration of peripheral nerves.
4. Focal degeneration of striated muscle.
5. Pronounced central and midzone degeneration of the liver. (This was similar to liver changes previously described by Aring.[12])

Through the courtesy of Dr. A. Theodore Steegmann, Kansas City, Missouri, the necropsy study of the spinal cord in case 14 is presented. His conclusions were:

1. Degeneration of the peripheral nerves.
2. Vacuolization of the cytoplasm of the anterior cornual cells.

Permission for necropsy was not obtained in the remaining fatal cases.

*Additional Cases.* Case 16 is included in this paper because it presents a case clinically identical with the Guillain-Barré syndrome associated with pregnancy,[56,74] and secondly because of the unusual subsequent development of papilledema.[4,36] This patient entered the Cleveland Clinic in October, 1942, thirteen days after she had been delivered of normal healthy twins. In the first three months of her pregnancy this patient had severe hyperemesis which resulted in a loss of 25 pounds. In the fourth month of pregnancy she developed a left facial palsy which lasted ten days. After the fourth month of pregnancy she noted weakness in her legs accompanied by numbness and soreness of her legs. The weakness progressed until the patient was unable to walk at term, and she also noted tingling and stiffness of her hands with mild clumsiness. Examination revealed loss of all deep reflexes and diminished sensation from the hips distally. Lumbar puncture revealed an initial pressure of 250 mm. of water with normal dynamics and xanthochromic fluid containing a total protein of 470 mg. percent and 2 cells per cu. mm. A tentative diagnosis of the Guillain-Barré syndrome was made and the patient discharged on supportive therapy. This same patient was admitted to the Johns Hopkins Hospital in Baltimore in January, 1943, unimproved, at which time she had bilateral papilledema and a cerebrospinal fluid pressure of over 600 mm. of water. Total protein was 420 mg. percent. An air injection was done and the ventricles were found to be normal. A right subtemporal decompression was carried out. At last report the patient had shown no improvement. Ford and Walsh have reported this case previously.[4]

Cases 18 and 19 are presented here as examples of the clinically identical picture seen in diabetes mellitus.

## CONCLUSION

From the foregoing discussion it is evident that a syndrome exists, of unknown etiology, which is characterized by polyneuritis, often with facial diplegia, usually presents albumino-cytologic dissociation of the cerebrospinal fluid, with a favorable prognosis in the majority of cases.

This syndrome is seen both as a primary condition and in association with diabetes, syphilis, diphtheria, serum sickness, fever therapy, pregnancy, exposure to mustard gas, and other conditions. The precise nature is obscure both in its primary and associated form. No recognized factors in any case of diphtheria, diabetes, syphilis, and the like, precede or prognosticate the development of this syndrome.

For the sake of clarification of terminology and out of deference to the men who first pointed out albumino-cytologic dissociation with polyneuritis, it is suggested that all cases of the aforementioned type, whether of the primary or the associated variety, be grouped under the title of the Guillain-Barré syndrome pending the time when the precise nature and etiology of this complex are understood.

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# Technic of Retraining in Psychotherapy*

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For convenience we think of psychotherapy in terms of four stages: ventilation, abreaction, insight and retraining. Actually, in practice, psychotherapy is not performed in any such routine way. The stages constantly overlap and are interchanged in order. Although we may try to follow that pattern in principle we often find that even during the first interview we are forced to make interpretations and even some retraining suggestions. Also as late as the final interviews we may be giving insight and some abreaction may be occurring.

The important thing to keep in mind concerning these stages is that to cure the patient we must retrain him. The other steps are secondary to and assist in the retraining. Freud and Adler spoke of analytic treatment as being a process of reeducation and practically every contemporary psychotherapist agrees with this concept. Nevertheless, we still hear and read too often about the magical accomplishments in our field: of cures resulting from clever suggestions or brilliant interpretations; from revealing a "mother fixation," an "oedipus complex," a "castration complex," feelings of guilt, or the inferiority complex; from the mysteries of narcoanalysis and hypnosis; from the advocating of a vacation on a ranch, of an "affair" or of a divorce; but as Adler, Freud, Alexander and all serious psychotherapists have insisted such processes at best only remove symptoms and that to cure the patient we must retrain him.

How may we reliably retrain him? For years it has been embarrassing to workers in the mental health field to witness the bitter conflicts among the various schools of psychopathology. Today the situation is a much happier one. All groups are coming to a more nearly common meeting ground. The demands of Horney and the recent modifications of psychoanalytic technic by Alexander and French carry them close to the nonpsychoanalytic therapists. There remains one main difference in technic. The advanced psychoanalytic group departs from the procedures set out here mainly in their heavy dependence upon the development of a transference neurosis and the careful direction of it. Our approach is more active with the therapist participating more fully. This discourages strong transference. But just as many early disagreements have proved to be only variations of terminology and not real differences so I believe will this one soon resolve itself. As psychotherapists we should be more interested in sound therapy than in belonging to any particular school of psychopathology.

There is practically universal agreement that to retrain the patient we must enable him to solve his problems in the light of a social community. Inasmuch as personality is the way one does things, retraining is actually a process of changing the personality. We help the patient to change his personality by showing him how he made it in the first place, and then, how to remake it using much the same method.

How did he, in his childhood, make his personality? We see young children at work selecting personalities every day. We see them, by trial-and-error, fumble at solutions to the problems which confront them each day. Mostly we see them trying to get possessions, attention and love. When they hit upon a means of solving their daily problems which they feel to be successful they keep repeating the same method. Whatever method seems to each one most successful becomes his method, and that method endures to the grave unless changed by depth psychotherapy.

This personality—this problem-solving technic—is learned, finished and made permanent in his early years. It is important to bear in mind that he chose his problem-solving personality before he was even aware of the basic problems which that per-

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sonality would be called upon to solve, before he was aware of such all-important concepts as, community, social interest, and fellow man, concepts which are the very foundation of all his problems, the foundation of the reality which will confront him. Having thus made his personality selection, first of all, by chance, that is by trial-and-error, and secondly, before being aware of the basic problems which will be crucial in his adult years, it is not surprising that he usually makes important mistakes in his personality structure.

We begin to retrain him by pointing out these facts. In learning that luck, and lack of awareness of proper goals, has much to do with his personality mistakes, his pride is protected, and he feels freer to change his ways. Unless this is done he acts as if his personality were given to him by divine grace and hence he strives doggedly to protect it at all costs.

To effect this change in his personality we place before him adequate goals for living, to replace his neurotic one; we show him how to solve his hour to hour and day to day problems with these new goals as his objective. This is the backbone of the reeducation procedure in most cases. It is repeating the manner in which he built his personality in the first instance, except that this time it will not be by trial-and-error and he will have before him the essential community concepts necessary for its successful functioning. After all, the whole of one's life is but a series of discrete problem situations; problems of work, problems of love and marriage, and the problems of general community value. So by helping him to solve each situation, as it arises, with a socially useful goal as the objective he learns how to face the whole of reality which is comprised of these situations. We analyze with him the method he uses in each situation, day after day, and we make suggestions for improvement. In thus checking with him his methods of solving those problems, we are comparing his methods with our own. It is in order to be sure of the validity of this method that the therapist must first be treated himself.

As he discusses his daily problems with us, the patient complains of his difficulties, misfortunes, or mistreatment. First, last, and all through the treatment we point out to him that he is creating the very situation of which he complains most bitterly. We must demonstrate this to him beyond any doubt, and over and over again, in order to get him to alter his attitude. We point out the reason why he creates these troublesome situations, that it is his purpose to deliberately select the wrong solution. To do that we must understand his childhood and his unconscious.

Further evidence of this attitude of creating his own bad luck is to be found in what I have called the constancy of "feeling-tone." In early years, everyone becomes accustomed to feeling a certain way; he accustoms himself to a certain, definite "feeling-tone." This is an unconscious process. All he knows is that a number of unfortunate things happened to him in his childhood which "made" him, he thought, feel a certain way. He believes himself to be the passive recipient of that feeling-tone. It became "second nature" to him. It is his normal accepted way of feeling. All through life he struggles desperately, but unconsciously, to maintain exactly that same "feeling-tone."

At the end of the first or second treatment, we give the patient a comprehensive history form to fill out. It is sought to elicit from his history the setting of the present situation, the childhood origin of this situation, and its causes. This gives us a quick look into the family psychological set-up and enables us to show present identifications and negative transferences, especially as they apply to the recreating of the childhood problem. There is no use to urge a patient to improve his marital habits if he identifies his wife with his mother. Nor is it of much value even to explain this identification to him if we do not also show him why he makes the identification. We must show him that he does so, in order to recreate the childhood setting. Perhaps—and we must make very sure of it—he had always leaned upon his mother for help, or perhaps only her praise satisfied him, or perhaps she was so critical he lived in last-

ing fear of displeasing her. To maintain the childhood "feeling-tone" he must transfer the particular relationship, which we find he depended upon in his childhood, to his wife. Having selected a wife who resembled his mother psychologically, she will react to him as his mother did and thus his "feeling-tone" is maintained. He has done this all himself, albeit unconsciously, and it must be revealed to him. Every psychotherapist has seen this illustrated many times in the type of patient who suffers in spite of a certain nobility of character. Such patients were "model" children who got much appreciation for performing unpleasant and difficult tasks without complaint. "She was an angel" her parents would say. Now, she must have hardships in order to prove her "angel role." So she, unconsciously, makes hardships.

What is the relation between the recreating of the childhood situation and the maintenance of a constant "feeling-tone"? The maintaining of constancy of "feeling-tone" is the reason why the patient continually recreates today the situations of which he complains. It is in order to maintain that "feeling-tone" that he must perpetuate the type of situation which originally aroused it. We find, therefore, that it is the fixity of feelings which prevents changes in a patient's life pattern. It is this fixity of feelings which makes difficult and even defeats our therapeutic attempts. Hence in our treatment we must focus constantly upon this feeling control.

Since this feeling control is an unconscious process, we must explore the patient's unconscious to discover what that control is and how it operates. All through the treatment we must keep watch over the patient's unconscious reactions to our efforts at freeing him from that control. We must help him free his actions from that control. There are a number of ways of helping him to do this: A most valuable procedure is to teach him this pragmatic rule: "Wherever you are and whatever you are doing, first decide what is the problem confronting you; second, plan the best solution to the problem that you can; third, proceed to carry out that plan unwaveringly to its conclusion without any regard whatever for your feelings, that is, regardless of whether you may fail, or whether you feel you do no want to carry out the plan, or whether you fear you may not be liked for so doing. If you think it is the best plan for solving the problem follow the plan regardless of your feeling." This apparently simple procedure, as an adjunct, has phenomenal results in freeing a person's actions from his rigid feeling control. There has been much nonsense written about the patient's inability to make changes contrary to his feelings. That is because many therapists formerly believed that one must do what he feels like doing. Casanova evidently lived according to the principle of doing what he felt like doing, but without the type of success and happiness most patients desire.

The basis of the retraining, then, is to place before the patient community-minded goals, and to help him to learn methods of solving his daily problems with those goals as his objective, and to point out to him continually his neurotic tendency to recreate his childhood situation in order to maintain his constancy of "feeling-tone." Once he really understands that, the reconstellation of his personality can proceed rapidly and deeply.

To perform adequately the retraining process, to make it more than soothsaying, it is necessary to reveal to the patient his unconscious. The unconscious is the home of feeling-controlled living and feeling-controlled living is compulsive living.

Any deep therapy must work at the unconscious level, and nothing but deep therapy can reconstellate the personality and hence be lasting. By knowing the unconscious origin and meaning of his symptoms and difficulties, we know exactly what to retrain and in what direction to train it.

How do we study the unconscious? Today we have many methods; some are rapid and accurate, some slower and more comprehensive. A fundamental fact to keep in mind is that the patient's unconscious determines his actions. Therefore by translating the meaning of his actions we quickly reach the unconscious. So we study the

meaning of everything he does. The first action we usually study in our search into the unconscious realms is his recollection of early memories. Out of the millions of things he did each year he recalls only a very few. These are recalled because they are part of the childhood situation which created his "feeling-tone." The patient whose early recollections all involve accidents, disaster and punishment, for example, is one who fears failure, and is one, therefore, who holds back with a so-called inferiority complex. He did not overcome his fears and so we know he hides behind fear. And today we will expect to see him evade his problems. A male homosexual recalled many incidents of misfortunes all caused by girls and women and meaning to him: "Stay away from women." His homosexuality is his method of evading women.

Dreams, of course, are wonderful unconscious pictures. It is wise to interpret dreams by use of word associations and free-associations. We all have had terrible shocks to our glib dream interpretations when the correct interpretation later became clear. Even when we are fairly certain of the interpretation it is well to encourage the patient to find the meaning in his own associations. It is more convincing to him and it often opens up new material of which we were not aware.

Free associations are basic in therapy. They take us quickly to unconscious material. A couch is not necessary. One needs only a quiet room and a comfortable chair. Necessary also is a therapist who is unhurried, friendly and permissive. Any point in a dream, or any puzzling situation in the past or present, is suitable for free association analysis. It may be used at any point in the therapy but is especially necessary when the patient brings to the treatment session no dreams or other problem material.

The thematic apperception test is at times a seemingly necessary source for revealing unconscious material.

The task of revealing and following the changes in the unconscious is made much easier by knowing in advance the psychodynamic basis of most neurotic situations. This we gain from the richness of our own clinical experience and from the constant reading of the present-day wealth of literature in clinical psychology and psychopathology.

For example, agorophobia is often caused by a powerful unconscious desire for extramarital exploration which is, at the same time, vigorously objected to by the super-ego. The resulting anxiety symptoms enable the agorophobic to compel someone, usually his wife, to accompany him whenever he leaves the house. This gives him protection from his libidinal desire. The blushing patient is often one who unconsciously desires sexual experience. Such experience, however, is abhorrent to her super-ego. The blushing tends to keep the patient at a safe distance from temptation. Migrain and severe headaches often indicate deep-seated and bitter resentment. Often the headache helps the patient to avoid, in part, the affronting situation, the unwelcome demands upon her, which she is not willing to refuse, consciously. This is frequently seen in the housewife with a strong super-ego who does every task her exacting husband demands because her super-ego will not permit her to refuse. However, when the headache appears she is freed from many of his demands without having to refuse.

As the patient redirects his problem-solving toward correct objectives we can observe the change in his unconscious. All of its manifestations change. His dreams change, different early memories are recalled, and his basic "feeling-tone" changes to one compatible with success and happiness.

Thus our modern technics in the practise of psychotherapy permit rapid changes in the personality which are entirely permanent. We are able today to reconstellate a personality in weeks or months that previously required years. Today our therapy is no longer prohibitive in price because it requires but twice-weekly visits, from one to six months, rather than daily visits from two to six years as it did formerly. The rapidity of technic, the control of transference, and the ease of unconscious checking, make our results more completely successful.

# Recognition of and Distinction between Post-traumatic and Psychophysical Headaches

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Frequent uncertainty is experienced in arriving at a diagnostic determination of divers complaints of headache. Without entering into a discussion of the causation of the many types of headache states, the two most commonplace types of cephalalgia here dealt with, are:

1) Headache as a resultant of head trauma.
2) Headache as a principal attribute of the various neuroses.

1) Frequently, following general brain concussion (which contemplates no vault fracture), loss of consciousness may supervene. and may be only for a moment, or, the unconscious state may extend for a more lengthy though temporary period. Upon return to the state of awareness, the characteristic and frequently protracted sequel is cephalalgia. It is usually complained of as being persistent over the occiput with the pain radiating down the neck. When it occurs, this troublesome symptom may be accompanied by a varying gradation of clouding of retentivity together with a diminution of mental activity. The onset of this syndrome may be immediate or may supervene the trauma following a lapse extending from a few days to weeks after the original trauma was suffered.

Electroencephalographic studies made directly subsequent to the accident, or air studies made at a later date, may reveal pathologies determinable as (a) concussion and (b) brain damage respectively. These techniques are of indispensable value when it is to be borne in mind that one is confronted diagnostically with subjective symptomatology which is frequently very bizarre and disordered. Here the credibility, as well as the incentive of the patient play an important role in the already involved task of differentiation between post-concussion syndrome and psychologic shading of symptomatology, when the latter may be effected for pecuniary gain.

Patients who have suffered from no prolonged lapse of the conscious state after a head injury, and who have not manifested the classical signs of traumatic cerebral compression, viz.: an increasing coma after lucidity, focal fits indicative of extradural hemorrhage, unilateral Babinski and contralateral hemiplegia, etc., at the time of the accident, have chances which are favorable for return to health. Not infrequently, however, the recovered patient will exhibit the troublesome syndrome of lassitude, poverty of intensive thought, coupled with headache, ease of excitability and impatience.

This will usually set in at any time within about two months following a head injury, else, in the absence of symptoms, recovery may be considered uncomplicated. Frequently however, there remains the syndrome of headache which is paroxysmal and bounding, together with vertigo upon exertion or postural alteration, as well as photophobia and querulousness. This syndrome is an apparent interaction of psychological and physical elements, particularly where these after-effects of head injury may be bound up with compensability predicated upon the patient's resolve to affix liability therefor against some one.

In injuries where actual brain damage has occurred despite no frank dissolution in scalp or skull continuity, the following pathology must always be borne in mind: From the clinical standpoint, histopathologic alterations do not always manifest a counterpart of severity of the head trauma suffered. The medulla seems to bear the greatest brunt in blows applied to the head. In other instances we may be confronted with contre-coup lesions of the (opposite) cortex in the shape of epidural and subarachnoid bleeding. There are many situations where no damage is apparent at the site of the blow, although bleeding has occurred in such locations as pons, basal ganglia and mesencephalon. Repair of resulting softening by glial formation will take place and nerve

cell loss may also be encountered. The long range result in many of these instances is encephalopathy due to the prior hemorrhages, upon which were superimposed the meningeal cicatrices, degenerative changes and glial tissue formation.

The exceptions to this usual and more common symptom mechanism following head trauma, may be the ushering in of unpredictable latent symptoms of acute concussion. These are characterized by the onset of a prolonged unconsciousness, impairment of medullary function, loss of superficial and deep reflexes, flaccidity, dilatation of the pupils, slow pulse, shallow respiration and drop in blood pressure. As the treatment of the latter is not within the scope of this discourse, the reader is referred to appropriate texts upon that subject.

2) The headache of the neurotic is characterized by subjective sensations of head pains, their usual description conveying no particularity of location. Neurotic patients will also recount sensations of a constriction about the head, or pressure upon it. In the more *severe anxiety reaction* states such neurotic patients will describe sensations which they characterize as though a spike were being driven into the skull usually subjectively allocated to the parietal and temporal sites. The *hysteric,* on the other hand, will, in addition, complain of a soreness of the scalp to the touch and of inability to obtain relief through any form of analgesic therapy. This is because the pain is fancied and a creation of the mind. As such, it manifestly cannot respond to medication. In conjunction with the above enumerated head symptoms, these patients frequently and unwittingly assist in the diagnosis by the presentation of a train of fantastic bodily symptoms. These are evanescent and have reference to all bodily parts and to sundry disfunctions. The general psychogenic demeanor of these patients is bizarre and may take on innumerable forms of reaction. To mention but a few; this type of patient may exhibit anxiety, excessive joviality, depression, belligerence, histrionics, loquacity, bodily preoccupation, unprovoked weeping, at times even anaesthesias of bodily parts not delimited by typical nerve distribution, or paralyses of limbs out of proportion to the severity of an injury.

Although this discourse does not include the subject of specific therapy, appropriate determination of the particular neurosis responsible for the non-organic headache will necessarily aid the therapist in his approach to the rationale of treatment.

The psychopathology of headache as a syndrome, when found in a patient suffering from *conversion hysteria,* is a precise expression of his mental imageries and desires into corporeal representation.

In the headache of the *anxiety reaction* state however, the pain expresses the physiological functions attendant upon anxiety. The tension of the latter is reflected in headache which is preceded by sudden involuntary contraction of the muscles underlying the integument of the head, as well as of the neck. This phenomenon is the outcome of an overloaded nervous system with stimuli to which the individual cannot convey sufficient outward responses.

There are a great many types of headaches where the definite etiology appears abstruse. It is a symptom complex referable and ascribable to many conditions, both organic and functional. In an endeavor to evaluate the diversified symptomatology, these diagnostic guide-posts are offered to clarify the two most frequently encountered patterns of headache states presented to us in our work-a-day obligations to both the ambulant and the bed patient.

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# Neurological Clinical Pathological Conference

of the
Cincinnati General Hospital
CHARLES D. ARING, M.D., *Editor*

CASE NO. 227709

## PRESENTATION

A 37 year old negro was admitted to the Psychiatric Service on July 9, 1947 because of strange behavior during the previous two weeks; he repeatedly filled a glass with water and poured it down the sink; he denied that he had ever seen a radio which he had owned for ten years; he thought he was back at his mother's home in Kentucky, and on several occasions he had defecated on the floor and urinated in bed believing he was in the bathroom. The night before admission to the hospital he had poured water into a clock explaining that it needed a drink.

Further information obtained from patient's wife indicated that he had had rheumatic fever at 14 years of age but had been well until 1936, at which time he developed "intestinal grippe" and was told by a physician that he had heart trouble and hypertension. He was advised to get an easy job. In 1942 he was rejected by the military service because of his heart. Approximately one year prior to admission he began to drink heavily. Four months before entry in the hospital he began to complain of bilateral parietal headache and shortly afterward was noted to be somewhat seclusive. After returning from his mother's home in May 1947, his wife observed him to be forgetful, careless about his clothes and person and to sleep more than previously. He exhibited increased sex drive. In June 1947, he ceased drinking on the advice of his physician and seemingly simultaneously developed pain in back of his head and neck, complaining of stiff neck, anorexia and loss of sexual desire.

The patient was unable to give an adequate or reliable history. He complained only of drowsiness during the previous month, denied the consumption of large amounts of alcohol, and stated that he had a job at the University Club and suspected his friends of plotting to get the job from him. He said that he had had a urethral discharge at three periods, a lesion on his penis during his youth and that he had received treatment for syphilis three months previously at Cincinnati General Hospital (however, no such record could be found); and his statements were judged to be a reflection of his mental condition.

On examination the patient displayed marked memory defects; he was disoriented as to time and place and was at times apathetic, at times somewhat euphoric. He showed no evidence of injury. The heart was enlarged somewhat to the left; and there was a soft systolic murmur and a loud diastolic murmur heard best near sternum and transmitted to the base and apex. There were no abnormal findings on neurological examination.

On the day following admission (July 10) he was confused but was allowed to walk about the ward whence he urinated on the floor. After a day during which he evinced much confusion, he developed generalized convulsions at 5:30 p.m., which continued in the typical fashion of status epilepticus for 50 minutes, and were controlled only after the administration of 8 cc. of paraldehyde intramuscularly.

He was resting comfortably after the medication when at 7:30 p.m. he developed Cheyne-Stokes respiration. The heart rate rose from normal to 200 per minute, the pupils became dilated and fixed; the corneal and deep reflexes were not obtainable. He expired 30 hours after he entered the receiving ward.

*Laboratory Data:* The temperature and respiratory rate were normal up to the time of the convulsions; his pulse rate descended from 86 at the time of admission to 60 twenty-four hours later. The blood pressure was 120/60. Blood count, hemoglobin determination and a blood Kahn were normal.

A lumbar puncture done at 4:00 p.m. on

July 10th revealed an initial pressure of 340 mm of water with good relaxation. After the removal of 13 cc the pressure was found to be 300 mm water. The fluid was clear and contained 18 lymphocytes, 2 polymorphonuclear leukocytes and 3 red blood cells per c mm. The total protein was 58 mg%, the Wassermann and gold curve were negative.

## DIFFERENTIAL DIAGNOSIS

*Dr. Milton Rosenbaum:* There is little question as to the diagnosis, but I will discuss the case as though I were the physician who first met this man on admission to the hospital and followed him during his course.

The patient was a 37 year old negro who had had symptoms for four months. His principal symptoms took three forms; headaches, mental symptoms and in the last month of his illness he complained of stiffness in the neck and pain in the back of his head and neck. The mental symptoms were classically those associated with disease of the brain and consisted of memory defects, personality change, apathy, drowsiness, and more acute mental symptoms (delirium) for the two weeks before entry.

Repeatedly filling a glass with water and pouring it down the sink, represents perseveration of behavior which is not unusual in brain disease. The fact that his mental symptoms became aggravated in the few weeks would make one think that the lesion responsible for the initial mental symptoms was aggravated or that possibly a new factor was operating. I believe that the factor responsible for the aggravation of the clinical picture was associated with increasing intracranial pressure resulting from the original brain disease. From his presenting symptoms a specific diagnosis could not be made. The only localizing symptom which he presented had to do with the stiffness and pain in the back of his neck which reminds one of something operating to create meningeal reaction.

Because of the lack of localizing neurological signs the patient was admitted to the Psychiatric Service, and there is little question but that the diagnosis entertained at that time was dementia paralytica. There was certainly justification for such a suspicion, especially since the neurological examination was normal as was the physical examination except for the evidence of rheumatic heart disease. One might have felt a little more secure in entertaining the diagnosis of dementia paralytica, if there had been pupillary abnormality and some of the other neurological signs usually associated with this disease. It must be remembered that in about 10 to 20 percent of paretics there is no pupillary or other neurologic abnormality.

Up to this point, I would entertain a diagnosis of organic brain disease, and breaking this down would consider paresis, some type of chronic meningitis such as tuberculosis or torula, and neoplasm. The next step in determining the diagnosis at the bedside would be an analysis of the cerebrospinal fluid. The probabilities are that when the spinal puncture was performed in the hospital, the diagnosis was considered to be paresis. The initial pressure was noted to be 340 mm of water, and in spite of this finding, 13 cc. of cerebrospinal fluid (probably more because of leakage) was drained off, and the final pressure was left at 300 mm. of water. The removal of such a large amount of fluid in the presence of increased intracranial pressure was a mistake in judgment.

As soon as the physician who performed the puncture saw how high the pressure was he should have immediately suspected the presence of brain tumor, and then, should have carefully withdrawn a few cubic centimeters of cerebrospinal fluid for cell count and Wassermann determination. It is my belief that with the proper precautions a spinal puncture can be performed safely in the presence of increased intracranial pressure. Naturally if the diagnosis of a brain tumor is perfectly obvious, there is no reason to perform this test. In this case such a diagnosis was not obvious, and it was quite proper to perform a tap.

One must realize that the physician is often faced with a patient who presents the signs and symptoms of increased intracranial pressure which is due to diseases other

than brain tumor. I personally remember several patients operated on for brain tumors who were actually suffering from acute luetic meningitis. Had spinal puncture and a cell count and Wassermann been performed, these patients would have been spared surgery. However, in our case the Wassermann was negative and the cerebrospinal fluid findings together with the clinical picture make the diagnosis of a brain tumor.

The cerebrospinal fluid dynamics in this case are of enough interest to warrant a few comments. Where the cerebrospinal fluid pressure is high due to a brain tumor, a rapid and marked fall in pressure usually follows the removal of a small amount of fluid. In those instances where pressure is high due to an accumulation of fluid with an internal hydrocephalus as in meningitis, it is necessary to remove large quantities of fluid to effect an appreciable drop in pressure. The Ayala index, which is described in detail in the book by Merritt and Fremont-Smith[1] on the cerebrospinal fluid, is a formula which may help to determine whether or not increased intracranial pressure is due to space consuming lesion or to an excess of fluid within the ventricular and subarachnoid spaces. One possible explanation for the peculiar dynamics in this case is that this patient had an excess of cerebrospinal fluid because he was suffering from an aseptic meningitis. The evidence for this is the mild pleocytosis in the cerebrospinal fluid and the clinical indications of meningeal reaction, namely pain and stiffness of the neck. An aseptic meningitis may have been due to encroachment of the neoplasm on the ventricles, the walls of which react to the presence of the foreign body (tumor) with sterile meningitis. Cases have been described in which rather severe forms of aseptic meningeal reaction have occurred with tumor.

Within an hour and a half after lumbar puncture the patient suddenly developed Cheyne-Stokes respiration and within a short time he expired with signs indicating brain stem involvement.

An important question to be considered is whether or not the death bore any relationship to the lumbar puncture. One must remember that patients with brain tumors not infrequently die unexpectedly, but here I believe the course of events make one strongly suspicious of a causal connection between the lumbar puncture and death. One might explain the death on the basis of medullary herniation through the foramen magnum or of a brain stem herniation producing acute vascular lesions in the brain stem. Since the patient did not have a posterior fossa tumor, it would be more likely that the cause of death was related to an acute interference with the blood supply of vital centers in the mid-brain as happens in brain stem herniation. I also believe that the sudden shift in intracranial structures and pressure may have been responsible for status epilepticus since the patient had suffered no previous convulsion, although one must remember that this might be a coincidence and that convulsive seizures are common in brain tumors.

My final point has to do with localization. It has been taken for granted by most physicians and many neurologists that the presence of mental symptoms is pathognomonic of frontal lobe disease. However, I wish to emphasize that mental symptoms in themselves are of little or no localizing value and may be associated with a brain tumor in any part of the central nervous system. In many instances, these symptoms simply reflect pressure changes and interruption of large and important pathways in the central nervous system. To further emphasize this, I should like to read a passage from Wilson[2]:

> In this connexion, more perhaps than in any other, the fallacies of the clinico-anatomical method stand nakedly revealed. Almost any textbook taken at random will be found, e.g. to allude to the "mental symptoms" of frontal tumour; but no idea that it is impossible to account for "positive" symptoms (to which class many of them belong) by lesions destroying cerebral tissue seems to be entertained. Nor does the intricate nature of cortical interaction, part upon part, appear to be considered. For these and other reasons most analyses and allocations of "mental syndromes" accompanying cerebral tumours are worthless.
>
> Since the presence of new growth is disclosed by the general neural complex of headache, giddiness, vomiting, and papilloedema, it seems equally natural to seek a general mental syndrome expressive of interference with the functions of the psyche. None has hitherto been found. Seldom, indeed, does the chance arise of assessing the patient's personality and mental constitution ere tumour symp-

toms appear, yet on them will depend in no little degree the reactions that he shows. Mental symptoms coincident with brain tumour may spring from the soil as a purely indirect sequel of the growth.

I would localize this neoplasm in the frontal lobe mainly because of the absence of focal neurological findings, and because of the rapid course of this man's illness. I believe the lesion was a large and rapidly growing glioma of the frontal lobes.

## DISCUSSION OF PATHOLOGY

*Dr. I. Mark Scheinker:* On gross examination there was considerable increase in bulk of the right frontal lobe and generalized flattening of the gyri especially noticeable over the anterior half of the brain. There was marked herniation and shift toward the left of the mesial face of the right hemisphere anteriorly.

Sections through both frontal lobes disclosed a diffusely infiltrating neoplastic growth displaying multiple foci of old and recent hemorrhage, cytic cavities, and areas of softening. In sections through the anterior portion of corpus callosum, there was a diffuse spreading of the tumor toward the centrum semiovale of the left hemisphere. There was swelling of the white matter of the right hemisphere and compression of the right lateral ventricle. The third ventricle showed a slight degree of compression and shift toward the left.

Sections through the mid brain revealed coalescent hemorrhages involving the median raphe and the red nucleus grossly.

The microscopic examination of sections taken from the frontal lobe neoplasm revealed findings characteristic of glioblastoma multiforme. There were numerous thrombosed veins and large areas of tissue necrosis. In addition there was marked vascular proliferation at the periphery of the neoplasm. The newly formed capillaries displayed proliferation and degeneration of the endothelial cells. Numerous mitotic figures were present.

Sections taken from the white matter of the right hemisphere displayed far advanced swelling with the histologic findings characteristic of cerebral liquefaction.

Sections taken from the mid brain revealed a tremendous congestion and stasis of the veins and capillaries associated with numerous perivenous hemorrhages. In addition there were several focal areas of tissue liquefaction.

This case illustrates the absence of focal neurological signs in the presence of a widespread neoplasm involving very nearly the entire right frontal lobe, the anterior portion of the corpus callosum and a small portion of the left frontal lobe; and also mental disturbances compatible with those described in cases of frontal lobe neoplasm particularly by the Germans. The evaluation of mental symptoms in the localization of cerebral neoplasms has been a matter of controversy for many years; a discussion of this problem is beyond the scope of this presentation. Whether there is a mental syndrome characteristic of frontal lobe lesion has neither been established or disproved in my estimation.

It is of interest that despite the presence of clinical and laboratory signs of meningitis (stiff neck, mild pleocytosis of the cerebrospinal fluid) there were no histologic signs of meningitis.

*Clinical Diagnosis*
*(Before Lumbar Puncture)*

Dementia paralytica.

Rheumatic heart disease.

*Dr. Rosenbaum's Diagnosis*

Glioma of the frontal lobes.

Aseptic meningeal related to encroachment of the tumor on the ventricular walls.

*Anatomical Diagnosis*

Glioblastoma multiforme of the right frontal lobe.

Cerebral swelling (stage of liquefaction).

Transtentorial brain stem herniation associated with numerous perivenous hemorrhages.

Mild chronic rheumatic mitral valvulitis.

Luetic aortitis.

Marked right atrial and ventricular dilatation.

*Dr. Charles D. Aring:* In the written discussion handed me by the students there was evinced considerable concern about the absence of papilledema in the face of such

high intracranial pressure. Those of us who see many neurological patients are not surprised by this combination of events since we have witnessed it often enough. I would refer you to the best discussion of the subject that I know about; namely that of Lauber on "The formation of papilledema" which appeared in 1935 in the ARCHIVES OF OPHTHALMOLOGY (13: 733-743).

1. Merritt, H. H., Fremont-Smith, F.: The Cerebrospinal Fluid, *W. B. Saunders Co.*, Philadelphia, 1938, pp. 333.
2. Wilson: "Neurology," *Williams and Wilkins*, Baltimore, 1941, pp. 1838. Volume 2, page 1224.

EDITOR'S NOTE: These conferences are conducted under the direction of Charles D. Aring, M.D., Professor of Neurology, assisted in preparation and execution by I. Mark Scheinker, M.D., Assistant Professor of Neuropathology, and Howard Krouse, Assistant in Neurology, University of Cincinnati College of Medicine.

# Neuro-Dermatitis From A Psychosomatic Viewpoint

(Report of Three Cases)

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Neurodermatitis has received little attention from psychiatrists and the few investigations reported in the literature consist of scattered and individualized case reports. The study of patients with skin disease offers an excellent opportunity for controlled psychosomatic observation since the lesions are readily observable and any changes which may occur can be correlated with the patient's emotional state. Neurodermatitis was originally considered to be due to some disturbance along the course of the nerves but now it is thought to refer to the dysfunction of the sympathetic nervous system and emotional background of the patient as being etiological agents in the production of the skin lesions. The skin lesions vary in appearance usually starting as a papular rash distributed over the face, neck, chest and flexor surfaces of the extremities always in areas accessible to the hand. The skin may become ichthyotic and thick as though covered with a layer of shellac. Intense itching is a uniform symptom present in all patients. Not all physicians agree on the etiologic relationship of emotional disturbances to the skin lesion. There are a respectable number who still consider the lesion due to some type of allergic manifestation. The following cases are all patients presenting the skin lesions rather typical of neurodermatitis and the psychopathologic picture present is introduced in each case.

*Case No. 1* was a twenty year old single girl who, seven years previously, had begun to have "itchy" areas over her face that would last from one to ten days and recur once or twice per month. The periods became more frequent and she became more annoyed. After a disappointing love affair one and one-half years before admission to Galveston State Psychopathic Hospital, the "itching" areas that had developed into a rash became generalized and she became asocial and discouraged. She was an angular asthenic appearing lady of dull-normal intelligence; of Louisiana French descent whose mother died when the patient was five years of age. Her father remarried and the stepmother beat her frequently; she was greatly frightened by her stepmother's behavior. At times she was locked out of the house and was refused food. Finally, when she was in her eleventh year, her stepmother threatened to kill her and the patient left home to live with an older sister. She was in the third grade and dropped out of school because no one compelled her to attend. She resided alternately with two sisters adapting herself to housework. The sisters fed and clothed her well enough but they never hesitated to remind her even in the presence of strangers that they were supporting her and that she was doing nothing to support herself.

At thirteen the patient began menstruating, a phenomenon for which she had been poorly prepared. It was shortly after this that the first red area appeared on her face. She was self-conscious about her facial rash. For six months she worked in a laundry and enjoyed the work very much but she was

forced to quit this job on account of the generalized condition that developed a year and a half prior to admission. Although a devout Catholic, she had never taken her troubles to the priest because she felt that he was old and could not understand her point of view. For over a year she had not even attended church because of the rash. Her only true love affair was that mentioned. She denied any sexual experience, however. A third sister, a cousin, and her father accused her of having contracted venereal disease, presumably syphilis, which accounted for the rash. Shortly after these accusations she terminated her engagement and subsequently developed the generalized body rash.

Unfortunately adequate rapport was never established and getting the case history was like pulling teeth. No sooner was there sufficient transference toward her admitting physician than he was shifted to a different service. A flare-up of symptoms was noted when there was a change in physicians. Aeration slowly expressed more material; reassurance and suggestion was offered routinely. Attempts to re-educate and offer distributive analysis had to be done in simple terms because of dull-normal intellect and partial illiteracy. The case was studied routinely in conjunction with the dermatology service. Excerpts from their reports read as follows:

"The patient has had chronic recurring erythematous lesions of the skin. These began with an irregular area of redness on the cheek which disappeared spontaneously only to recur and progress to cover the entire face. It would seem that the lesions have never been too acute and when the skin is not covered with an emollient it becomes dry and scaley. Later the dermatitis extended to cover the trunk and sides. Only within the last six months have the legs become involved. At the present time the dermatitis is most severe on the leg. She has had numerous types of local medication but has found that plain vaseline gives the most relief. When the skin is dry it itches. There is a widespread very mild erythema of the skin. This is more pronounced on the legs around the ankles and feet. It is rather diffuse in nature and rather difficult to detect because of its faintness. The fingernails are of interest in that they are marked by discrete depressions, particularly near the lower margin. These, no doubt, are the result of interference with keratonizing processes due to the involvement of the skin at the matrix of the nail. The nature of the dermatitis is like that we have formerly observed in patients who were involved in some emotional conflict. Impression: 1. Neurodermatitis. 2. Psychoneuroses."

Long and frequent discussions were held. She remained somewhat evasive and distracted. She spoke without expression and in monosyllables. A number of situational factors were noted: poor social existence, inadequate outlets, poor family guidance, financial insecurity, false sexual accusations by father, hesitancy about forming emotional attachments toward her environment, and most of all, hostility toward her father and stepmother. During the interviews it was interesting to find the sudden skin reactions while discussing her possible conflicts. At these times the mild erythema would become more noticeable, her face in particular would become reddened and the "itching" would become more severe. All of this might occur during a short interview lasting only a few minutes. There was slow and gradual, but insufficient, improvement during her four months hospital stay, after which time she was discharged.

*Case No. 2* was a 46 year old, white, married male, whose complaint on admission was "skin rash, headaches and intestines draw up in knots." A dermatological report on admission to the hospital read, "the skin on the palmer surfaces of both hands is hyperkeratotic with hyperemia, excoriations and crusting. The skin is cracked and swollen on both lower legs and feet. There are maculopapular eruptions on erythematous bases. Excoriations are present and there is considerable evidence of scratching. He has somewhat the same pathology on the abdominal and inguinal regions. Impression: Neurodermatitis." Fourteen years prior, he had a leg rash with intense itching and the lesions remained with frequent exacerbation. He attributed the rash to poor bowel elimination. The itching had become more intense, interfering with his sleep, making him more irritable and thus created a "nervous exhaustion." He was first admitted on a dermatological service and then transferred to psychiatric care. A number of possible situational factors were elicited

during his clinical course such as: domineering and extremely religious family; excessive ambitious drives; the patient was told by his mother that he might never have a child and he was always considered by himself as being only "part man" since one of his testicles never descended, as a result, he may have had even more of a high ambitious level in order to prove that he was a man; a strong attachment to his mother; incest with sister as a child; unsatisfactory heterosexual intercourse as an early adult; loss of a very good job as an executive and demotion to a worker in overalls with men considered to be below his class; married to a widow who proved a domineering wife; difficulty in adjusting himself as a stepfather and a husband; wife's hysterectomy which had created in his mind an impression that she no longer desired sexual intercourse; concern over frequent masturbation in earlier years; and recognition of his own financial gain by the death of his stepson during the war. With a certain amount of rapport and confidence established on admission, there followed: frequent interviews, evaluation, hypnosis, sodium amytal interviews, attempts toward analytical understanding and supportive measures. It was during this therapy that the possible situational factors were elicited. The patient accepted his problem as psychogenic and elaborated in great detail on a conscious level in an effort to bring up all things that might have created his difficulty. The itching of his hand subsided. There was less concern with his bowel movements. Although there were good days and bad days, there was progressive improvement both objectively and subjectively until discharge.

*Case No. 3* was a thirty-eight year old white male whose present illness began six years prior to admission as minute vesicles on the fingers of the right hand. The lesions later appeared on the left hand and both upper and lower extremities. The spread of the eruption to its admission distribution had been accompanied by severe itching; in fact, over certain areas it seemed that the itching preceded the visible changes in the skin. It was the opinion of the patient that much of the dermatitis was the result of scratching. The skin was very red and rough. The opinion of the dermatologist was that the patient had neurodermatitis. He worked irregularly because of the dermatitis. The course of disease was characterized by recurrent acute episodes when small blisters would appear, especially on the hand, and there had been severe itching followed by frantic scratching. During the period between these attacks the dermatitis subsided, almost disappeared. It was noted by the patient that when under emotional stress the dermatitis suddenly became worse. Once he had dermatitis of the forehead which he attributed to a hat band. Frequently he has been awakened by severe itching. He has consulted many physicians. The dermatitis had been attributed by some to hypersensitivity. He was once found sensitive to wheat, yeast, malt and coffee. When these were removed from the diet no apparent effect on the severity of the dermatitis was observed. After his marriage the patient was engaged in giving violin lessons to children. He gave this up because he found the pupils too irritating and did not provide sufficient income to support his family. After giving up this work he has never been happy in any of his occupations. His wife reported that when the patient was having difficulty with his work the dermatitis was most severe. Three years before admission the patient became infuriated with a building contractor following which there was an aggravation of the dermatitis. Five years prior to admission the patient had a vasectomy. At this time they had two children and his wife decided that she did not want anymore. The patient, a Catholic, stated that he "squared this with the priest's O.K." These flare-ups continued until about three weeks prior to admission when the dermatitis became worse as the wife threatened to leave him following a drinking bout. It was found during interviews that he complained about his two daughters worrying him by doing trivial things. It was also elicited that there was conflict between his parents when the patient was younger and they have been recently divorced. He was given frequent interviews during which it was felt that much was accomplished in the

way of ventilating material in relation to difficulties with his family and his conflicts over other matters. It appeared that the patient had been placed in an environment which he was inadequate to handle. Also that the pleasure gained by the diversion of his sexual and environmental troubles to his skin was such that the patient scratched in order to provide an emotional outlet for his conflicts. This "itch and scratch" or "sadomasochistic" trend seemed to exist not only in this case but in others as well.

Reports on psychosomatic medicine stress the fact that other psychoneurotic symptoms are present in these cases which is also the fact in the three cases reported. Hostile tendencies were the most marked feature present in all of these cases and there seems to be a definite correlation between events which evoked the feelings of anger and the exacerbation of the eruption on the patient. There were also symptoms of depression and in at least one case evidence of obsessive and compulsive symptoms. The exacerbation of the depressive feelings usually followed the anger and the eruption. In these patients there was a prominence of sexual maladjustment. strong feelings of hatred, fear and guilt, conflict over masturbation and in all instances some secondary gain brought about by the illness. Appropriate local and systemic treatment was used supportively with considerable benefit. There was a lessening of the tension in the course of treatment which consisted of recreation, elimination of exposure to their environment brought about by hospitalization and accentuating the patient's interest in various recreations and hobbies. All the patients presented marked feelings of inadequacy, of being different from the group, having a great tendency to anticipate trouble. They all had rigid moral and ethical standards accompanying their rather labial emotions. This required additional care in giving guidance of an active sort in order to get the patient to assume more and more the final resposibility for the decision of each individual situation which in turn called for more guidance. Each of these patients showed a tendency to take more than the usual amount of sleep, they were all asthenic, they all had rather labial vasomotor symptoms manifest by labial pulse rate, dermatographia, hypersensitive skin and they were also quite shy, sensitive and timid. Each of them had a great tendency to dwell on the past and to anticipate the future. In no case was the typical symptom formation suggesting hysteria or complete conversion reaction seen. The psychiatric approach was made on a broad basis and these patients all required considerably in the way of time and not in some type of analytic work desirable but in addition these patients require more than the usual degree of active guidance.

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